

HIV INFECTION

— Tested to Death —

A special report which draws together the mounting evidence that indicates HIV is not linked to AIDS.

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Virtually all of the scientific community has taken a germ-theory view of AIDS and accepted the theory that the Human Immunodeficiency Virus (HIV) is capable of causing a syndrome of ever-advancing immune-suppression from which you will eventually die.

However, a few lone heretics such as leading University of California Professor of Molecular Biology Peter Duesberg, and Australian biophysicist Eleni Papadopoulou-Eleopoulos, have offered exhaustively-referenced argument that HIV infection does not lead to AIDS and that the test is so inaccurate that it shouldn't be used. They have been vilified for their troubles, and Duesberg, named an outstanding investigator by the American Cancer Institute, has had government funding withdrawn.

If they are only half right, the implications are indeed chilling. By rallying around the HIV theory, we may be erroneously lumping together under the umbrella term "AIDS" some 25 disparate, previously-identified diseases acquired by a number of non-contagious means (see box, page 12). This, coupled with the evidence about the inaccuracy of the tests, means that we could be misdiagnosing as HIV-positive many thousands of basically healthy people and subjecting them to highly toxic, potentially lethal drugs, the side-effects of which are now indistinguishable from what we consider AIDS-related illness.

In a 76-page paper which Duesberg published in Britain (*Pharmacology and Therapeutics* (1992), 55:201-77), he systematically takes apart the theory that AIDS is caused by an infectious virus and that HIV is capable of the wholesale destruction claimed. Co-discoverer of HIV, Robert Gallo and others have based their theory of the HIV-AIDS link on purely circumstantial evidence: that HIV seems to be present in all patients who have AIDS. Nevertheless, as long ago as 1989, Luc Montagnier, the French co-discoverer of HIV, admitted: "HIV is not capable of causing the destruction to the immune system which is seen in people with AIDS."

Duesberg quotes the Institute of Medicine's statistics which show that no more than about 50 per cent of American AIDS patients have antibodies against HIV. Furthermore, the US Centers for Disease Control and Prevention in Atlanta has confirmed the existence of cases reported of "T-lymphocyte depletion in persons without evident HIV infection" (*JAMA*, 9 September 1992).

Every direct measurement of AIDS is incompatible with all the classical criteria for infectious disease, says Duesberg. First of all, very few cells are actually infected with HIV—the average is only one in 1,500 to 8,000 white blood cells in AIDS patients. In fact, many healthy HIV-carriers have 40 times more HIV-infected white blood cells than do AIDS patients. "Since on average only 0.1 per cent (1 out of 500 to 3,000) of T-cells are ever infected by HIV in AIDS patients, but at least three per cent of all T-cells are regenerated...during the two days it takes a retrovirus to infect a cell..., HIV could never kill enough T-cells to cause immunodeficiency," writes Duesberg. "Thus, even if HIV killed every infected T-cell...it could deplete T-cells only at 1/30th of their normal rate of regeneration.... The odds of this desultory rate of annihilation being able to topple your immune system," he says, "would be the same as those of a bicycle-rider trying to catch up with a jet airplane."

Furthermore, since 1985 when HIV was first detected, the number of Americans infected has remained at a constant one million—which tends to indicate a virus that has been long established in the population. And never in the history of man has infectious disease discriminated against most members of the population as this one supposedly has, preferring only homosexual men, drug users, haemophiliacs and Africans. Nor do we know of another virus that takes 10 years to incubate into disease.

Eleni Eleopoulos notes that about a quarter of the population of southern Japan has antibodies against the HIV virus, compared with one per cent of the population of the US. Nevertheless, at the time of writing, only 14 cases of AIDS had been reported in Japan—a figure that has not significantly increased (*Medical Hypotheses* (1988), 25:151-62).

One study, performed at St Mary's Hospital in London in the mid-eighties, demonstrated that even HIV-negative homosexual men had significantly reduced T- and B-cell activity compared with heterosexual controls. In fact, their immune systems were just as suppressed as those of symptomless HIV-positive homosexual men (*Clin. Exp. Immunol.* (1989), 75:7-11). This finding would seem to support the argument that elements in the modern homosexual lifestyle, independent of HIV infection, are responsible for immune-suppression.

Studies have found that less than one-third of patients with Kaposi's sarcoma, one of the main illnesses associated with AIDS among homosexuals, are HIV-positive. Researchers at the CDC now accept that KS, one of the original and most specific of AIDS-defining illnesses, is not caused directly or indirectly by HIV (*The Lancet* (1990), 1:123-8). Furthermore, for low-risk groups such as the wives of haemophiliacs, inadequate proof exists of infection. Since 1985, only 94 wives of the 15,000 HIV-positive haemophiliacs have supposedly developed AIDS-defining diseases. However, given the small number, and the fact that most of these women have died of age-related opportunistic infections

such as pneumonia, Duesberg argues that an association between them and HIV infection has not been established. In another study, of 41 wives of immunodeficient haemophiliacs, all the T-cell ratios of the women were normal (*JAMA* (1984), 251:1450-54).

The proof of HIV as the causation of AIDS entirely hinges on the idea that detection of an antibody response to the virus is proof of its actual presence. In other words, the assumption is that if your body has made antibodies specific to HIV, it must mean that a protein of the virus and, hence, the virus itself is present. This is so because the so-called AIDS tests cannot test for the presence of HIV, just the presence of antibodies to it—the usual sign that the body has fought off infection.

The HIV tests are themselves known to be highly erratic and unreliable. The enzyme-linked immunosorbent assay (ELISA) test is most frequently used to test your HIV status, and the Western Blot is used as a confirmation. What happens with ELISA is that a sample of the patient's blood is added to a mixture of proteins. It is assumed that if HIV antibodies are present in the

blood, they will react to the HIV proteins in the test. With the Western Blot, these HIV proteins are isolated in bands; when mixed with a blood sample, each protein band will show up if it has bound to an antibody.

The CDC considers a single ELISA test without any other confirmation, proof positive that you have HIV infection—hence, eventually, AIDS.

The ELISA test is notoriously unreliable; in Russia in 1990, out of 20,000 positive ELISA tests only 112 were confirmed using the Western Blot, notes Eleopoulos.

Furthermore, neither test is HIV-specific—both react to many other proteins caused by other diseases. For example, the protein p24, generally accepted to be proof of the existence of HIV, is found in all retroviruses that live in the body and do no harm. Dr

Gallo has stated repeatedly that p24 is not unique to HIV—(*Bio Tech*, June 1993). Hepatitis B and C, malaria, papilloma virus warts, glandular fever, tuberculosis, syphilis and leprosy are just a few of the conditions that are capable of producing biological false-positives in ELISA tests (*Nature* (1985), 317:395-403 and *The Lancet* (1989), 11:1023-25). In one study, antibodies to p24 were detected in one out of 150 healthy individuals, 13 per cent of randomly selected otherwise healthy patients with generalised papilloma virus warts, 24 per cent of patients with cutaneous T-cell lymphoma, and 41 per cent of patients with multiple sclerosis (*New England Journal of Medicine* (1988), 318:448-9).

What's the truth?

Professor Peter Duesberg (University of California at Berkeley) was the person who made the most noise as to the likelihood of HIV not being the cause of AIDS. He was followed soon after by Professor Robert Root Bernstein whose book, *Rethinking AIDS*, is a devastating indictment of the muddle and intrigue of AIDS politics and pseudo-science.

The debate has been documented by *The Sunday Times* (UK) science correspondent Neville Hodgkinson, whose 3 April 1994 article, "Conspiracy of Silence", highlighted scientific scepticism regarding the HIV=AIDS myth.

Here are quotes from some of his witnesses:

- "The 'HIV causes AIDS' dogma represents the grandest and perhaps the most morally destructive fraud that has ever been perpetuated on young men and women." — Dr Charles Thomas, former Harvard Professor of Biochemistry.

- "The way the HIV theory is being applied is unfalsifiable and therefore useless as a medical hypothesis. AIDS is the result of an enormous level of exposure to human viruses and bacteria." — Dr Kary Mullis, 1993 Nobel Prizewinner for Chemistry.

- "There are many people with AIDS but without HIV, and a great many people with HIV but without AIDS. The HIV+AIDS hypothesis is much too simple. Plausible alternative, testable causes...should become part of regular AIDS research." — Dr Hank Loman, Professor of Biophysical Chemistry, Free University of Amsterdam.

- "Evidence is rapidly accumulating that the original theory of HIV is not correct. It is not sufficient alone to cause the disease." — Dr Steven Jonas, Professor of Preventive Medicine, State University of New York.

(Source: *Int'l Journal of Alternative and Complementary Medicine*, August '94)

In a 1991 study it was noted: "In half of the cases in which the subject had a positive p24 test, the subject later had a negative test without taking any medications that would be expected to affect p24 antigen levels." The researchers concluded that the "test is clinically erratic and should be interpreted cautiously." (*Abstracts, VII International Conference on AIDS, Florence, Italy, 1991, vol. 1, p. 326.*)

The French government has recently withdrawn nine of the 30 HIV tests.

Western Blot, which is supposed to be the more accurate of the two, is no more specific than ELISA. Dr Max Essex of Harvard University's School of Public Health, a highly respected AIDS expert, found that the Western Blot gave a positive result to some 85 per cent of African patients found to be HIV-negative. Eventually they discovered that proteins from the leprosy germ—which infects millions of Africans—can show up as a false positive on both ELISA and Western Blot (as reported in *The Sunday Times*, 22 May 1994).

In one study of Venezuelan malaria patients, the rate of false positives with Western Blot was 25 to 41 per cent. This led the researchers to conclude that "HIV is not causing AIDS, even in the presence of the severe immunoregulatory disturbances characteristic of acute malaria." (*New England Journal of Medicine* (1986), 314:647.)

Eleni Eleopoulos and her cohorts argue that there has been no standard established to interpret what the individual strips on the Western Blot test actually mean. In the US, the Transfusion Safety Study Group submitted some 100 patient samples weekly for testing to three highly respected laboratories over three periods of several months. The TSS found extreme variations in band patterns of the same samples even at the same labs.

The lack of specificity of HIV-testing should be disturbing to all clinicians working with people deemed to be HIV-positive. Individuals belonging to the main AIDS 'risk' groups—gay men, drug users and haemophiliacs—are exposed to many foreign substances such as semen, drugs, blood transfusions and blood components, hepatitis, Epstein Barr virus and many other factors or diseases known to cause false positives. Other populations—such as Africans and drug users—exposed to a greater than normal amount of disease, also make many more antibodies than the rest of us and therefore are likely to throw up false positives. For

instance, Eleopoulos claims there is a strong association between blood transfusions and a positive HIV test.

In one study (*The Lancet* (1986), 1:1090-92), the amount of HIV antibody detected in ELISA tests was greatest immediately after blood transfusion and decreased between transfusions.

In another instance (*AIDS* (1988), 2:405-6), a volunteer was given six injections of donated HIV-negative blood at four-day intervals. After the first injection his HIV test was negative, but the signal of a positive antibody response increased with each transfusion.

Once a patient is shown to be HIV-positive, doctors persuade them of the importance of regularly testing their blood for abnormalities, particularly for a significant drop in the number of T-helper cells (CD4s), considered an indicator of the presence of major diseases associated with AIDS.

CD4 counts are practically meaningless as a measure of disease progression and can show a rise or a fall at any given point in the day. In many clinics throughout the world, 'significant' decline in CD4s is used as a marker for future prophylactic drug treatment. Two hundred T-cells per millilitre of blood is considered the point at which the patient will be told that without drugs like AZT, ddI or Septrin, their chances of acquiring one of the diseases associated with AIDS, such as *Pneumocystis carinii* pneumonia (PCP), are fairly high.

What, then, is AIDS?

If HIV isn't the cause of AIDS, what is? In Peter Duesberg's view, "Twenty-five previously known, and in part entirely unrelated diseases have been redefined as AIDS, provided they occur in the presence of HIV." (*Pharmacology and Therapeutics* (1992), 55:201-77.) In other words, if you've got tuberculosis without a positive ELISA test, you've got tuberculosis. If tuberculosis is diagnosed with a positive HIV test, you've got AIDS.

The heretical view is that the severe immune-suppression seen in full-blown AIDS is not caused by a germ, but by a variety of separate lifestyle risk factors peculiar to the various high-risk groups which have compromised health prior to the onset of the disease. Their common thread, believes Eleni Eleopoulos, is that they are all oxidising agents—that is, they cause unwanted "fires" in cells, as cancer does. These insults to the body only cause immune-suppression after a long window of time, just as cigarette-smoking takes years to lead to cancer.

The prevailing belief is that this immune-suppression is caused by:

- among male homosexuals, a high number of sexual partners, receptive anal intercourse and exposure to recreational drugs, especially nitrites. According to Eleni Eleopoulos, abundant studies show that immunosuppression and the development of Kaposi's sarcoma is related to the number of sexual partners and frequency of receptive anal intercourse; indeed, immune-suppression appears more often in anal sperm recipients (i.e., passive partners) but not in their sperm-donating partners. Furthermore, animal studies conclusively demonstrate that sperm is a strong immunosuppressive if it migrates to the general cells of the body. This is more possible with anal sex since, unlike the vagina with its thick lining which makes penetration of semen into the bloodstream unlikely, the rectum is separated from the bloodstream and lymphatic system by only a thin cell-wall, which is easily penetrated during anal intercourse (*Medical Hypotheses* (1988), 25:151-62).

- among haemophiliacs, not only by the condition itself, but also impurities in the blood-clotting agent Factor VIII. In one study, more non-HIV haemophiliacs demonstrated AIDS-related diseases than did those who were found to be HIV-positive (Gomperts, E. D., de Biasi, R. and De Vreker, R., *The Impact of Clotting Factor Concentrates on the Immune System in Individuals with Hemophilia*, Baxter Healthcare Corporation, Hyland Division, Glendale, California, 1991).

- among intravenous drug users using opiates, the immunosuppressing effects of drugs, which have been known since the 1970s.

- among recipients of transfusions, blood products and transfusion itself, which is known to be immunosuppressive, particularly when irradiated blood is used. One study demonstrated that the more blood a patient receives, the lower his number of T-cells (*British Journal of Haematology* (1985), 59:713).

- among babies, supposedly born to HIV mothers, drug addiction or the effects of drug addiction in the mother. Three-quarters of American AIDS babies are born to "crack mothers", says Duesberg. Other AIDS babies are blood transfusion recipients or haemophiliacs.

- among Africans, malnutrition and widely practised anal intercourse among heterosexuals. AIDS is a new name for old, indigenous diseases. Also, what we call an "AIDS epidemic" could be the result of wild inaccuracy of the AIDS test in Africa in which thousands of people with multiple antibodies show up as HIV-positive on ELISA tests. Unlike the US and European AIDS cases, African AIDS doesn't occur among homosexuals, drug users or haemophiliacs.

- among all AIDS patients, prophylactic drugs like AZT, which cause anaemia and lower blood cell count, causing "AIDS by prescription", Duesberg says.

Many studies have shown that the average T-cell count for a non-HIV tested person can vary from as low as 200 up to 2,000. There are many instances of people with fewer than 50 T-cells who remain perfectly healthy. In a recent BBC *File on Four* documentary, Professor Ian Weller, who coordinated the British arm of the Concorde AZT trial testing the drug on healthy HIV-positive volunteers, commented: "The thing we have to remember about CD4 (T-cell) counts is they are very variable. They can vary in an individual over the time of day...lower in the morning and higher in the evening. They can be affected by things that you do such as walking to the clinic, as opposed to riding a bike...the amount of sunshine can affect them. Smoking as well."

Another variable which can seriously affect the outcome of CD4 testing is the sheer inaccuracy of laboratory process. For that same programme, a volunteer elected to have blood taken for T-cell counting.

Two samples taken from the same vein at the same time with the same needle were sent to two different laboratories. The resulting CD4 counts varied by 33 per cent! Once an otherwise healthy patient has tested HIV-positive, has a low CD4 count and is exhibiting stress-related problems, virtually all doctors working in the field reach for the prescription pad and offer prophylaxis.

The idea is to give the patient a drug on the assumption that this just-in-case medication will stop the disease before it starts. In the case of the main anti-HIV drug AZT, this assumption was demolished with the recent publication in *The Lancet* of the Concorde Trial results, which showed that AZT was of no benefit to HIV-positive individuals who remained asymptomatic (*The Lancet*, 9 April 1994).

Besides there being no rationale for their use, a patient with no symptoms given these drugs begins to exhibit all the problems associated with the side-effects of the drugs—side-effects that bear an uncanny resemblance to the list of symptoms doctors describe in HIV infection or full-blown AIDS.

The Kamikaze Cell

As Duesberg and Eleopoulos point out, HIV is one of some 100 to 150 latent retroviruses in humans, all with the same genetic structure. In the early 1980s, both Robert Gallo from the US National Cancer Institute and Luc Montagnier from the Pasteur Institute individually isolated a retrovirus from homosexual patients believed to have AIDS. According to the original Gallo/Montagnier theory, this retrovirus is a virus whose own RNA (short genetic code instructing each cell how to reproduce) is transmuted by a particular enzyme (reverse transcriptase) into DNA, the long double helix of complete coded cell information. According to the single-cause theorists, HIV can 'break into' an immune system T-cell and attach itself to the cell's DNA, feeding off it. Once the cell replicates, the virus itself replicates, too. Once a patient has full-blown AIDS, the virus supposedly will have devoured these cells, leaving the body without defence to any sort of disease.

With all the giant holes in this theory, the AIDS 'revisionists', including co-discoverer of HIV Montagnier, have attempted to salvage the HIV hypothesis with the proposition that HIV needs one of a number of co-factors, such as mycoplasma and other viruses to induce cell death. Or, in some way unique to the history of infectious disease, HIV can talk the immune system into reacting against itself or committing suicide.

Duesberg points out that many retroviruses exist in every cell, not simply in a few, as HIV does. And most of the AIDS risk groups have a high number of antibodies to many human parasites which have been accumulated through high-risk behaviour (such as drug use), contaminated transfusions or high promiscuity. He and Eleni Eleopoulos propose that HIV may be an undistinguished "innocent microbial passenger". Hence, although HIV may be perinatally transmitted to babies, it could be harmless.

AIDS-defining illnesses

AIDS is usually characterised by a number of 'opportunistic' diseases, which can become active in a body whose immune system has been severely compromised. These include *Pneumocystis carinii* pneumonia (PCP) and candidiasis (both fungal parasites present in all humans), tuberculosis, toxoplasmosis, cytomegalovirus and herpes virus disease. In addition, AIDS patients can suffer from other illnesses not caused by immune-deficiency, including lymphoma and Kaposi's sarcoma (two forms of cancer), dementia and wasting disease. If AIDS were caused by a virus, it has a strange proclivity for giving certain groups certain types of diseases. For instance, Duesberg points out:

- American homosexuals have Kaposi's sarcoma 20 times more often than all other American AIDS patients. Less than one per cent of haemophiliacs and about the same percentage of Africans get KS. Studies have linked KS with anal intercourse and "poppers"—amyl nitrite.
- Intravenous drug users tend to get tuberculosis.
- "Crack" users get pneumonia, in addition to tuberculosis.
- Haemophiliacs favour opportunistic infections, three-quarters of which are fungal, plus viral pneumonia.
- Blood transfusion recipients get pneumonia.
- AIDS babies tend to get bacterial diseases and dementia.
- Ninety per cent of African AIDS patients, who are evenly divided among males and females (unlike the West), get fever, diarrhoea, tuberculosis and "slim disease"—all long-established diseases of the continent. Africans do not get PCP and candidiasis, even though both parasites exist in all humans including Africans.

A recent study concluded that prophylaxis with the antibiotic Septrin against PCP is far more likely to cause the patient to develop oral candidiasis, wasting syndrome, cytomegalovirus and *M. avium* complex disease—all commonly considered AIDS illnesses—than in patients who don't take the drug (*New England Journal of Medicine*, 23 December 1993).

The action of both this drug and AZT destroys or inhibits enterobacteria in the gut including *E. coli*, thus causing an overgrowth of *Candida* and other unwanted bacteria which cut off the body's ability to manufacture intrinsic factor required for the absorption of vitamin B12. The final symptoms of end-stage B12 deficiency are identical to the final stages of terminal AIDS.

Editor's note: Jody Wells, who was diagnosed as HIV-positive 11 years ago, remains healthy without the use of drugs. He is founder and editor of *Continuum*, a magazine for AIDS and HIV survivors. For more information, write to PO Box 2754, London NW10 8UF, United Kingdom. Phone: +44 (081) 961 1170.

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